

Lneonatal meningitis due to streptoccocus dysgalactiae subspecies equisimilis: a case report and literature review

Neonatal streptoccocal menengitis

Nuriye Tarakci¹, Fatma Hilal Yılmaz¹, Nazlı Dilay Gültekin¹, Hüseyin Altunhan¹, Rahmi Örs² ¹Department of Neonatology, Meram Medical Faculty, Necmettin Erbakan University, ²Medova Hospital, Konya, Turkey

Abstract

Streptococcus dysgalactiae subsp. equisimilis (SDSE) was firstly reported as a toxon among human streptococcal pathogens in the year 1996. Diseases caused by SDSE may vary from milder skin involvements including wound infection, erysipelas, and cellulitis to life-threatening clinical pictures as streptococcal toxic shock syndrome and necrotizing fasciitis. We describe a case of SDSE sepsis and meningitis in a 2-day-old newborn. He was referred to our intensive care unit (ICU) with fever, respiratory distress, seizures, peripheral cyanosis, and somnolence. SDSE grew out of the CSF and blood cultures obtained on admission to the ICU. SDSE cases reported in the literature are frequently older patients with an underlying disease. Also, SDSE may cause serious neonatal infections.

Keywords

Streptococcus Dysgalactiae Subspecies Equisimilis; Neonate; Meningitis

DOI: 10.4328/JCAM.5697 Received: 17.01.2018 Accepted: 14.02.2018 Publihed Online: 16.02.2018 Printed: 01.05.2018 J Clin Anal Med 2018;9(3): 254-6 Corresponding Author: Nuriye Tarakci, Department of Neonatology, Meram Medical Faculty, Necmettin Erbakan University, Konya 42080, Turkey. T.: +90 3322236000 F.: +90 3322236585 E-Mail: nuriyetarakci@hotmail.com

Introduction

Neonatal sepsis is an informed serious disease, and Group B streptococci (GBS) is the most common cause of neonatal sepsis. The use of prophylactic antimicrobials has decreased the incidence of GBS-related disease [1]. SDSE was firstly reported as a human pathogen in 1996 [2]. This microorganism has Lancefield Group C or G antigens and rarely an antigen. This microorganism causes beta hemolysis and exerts streptokinetic activity on human plasminogen and proteolytic activity on human fibrin. SDSE can be found in the oropharynx, gastrointestinal tract, genitourinary tract and as normal skin flora. Diseases caused by SDSE vary from mild skin infections, such as wound infections, erysipelas, and cellulitis to life-threatening infections, such as streptococcal toxic shock syndrome and necrotizing fasciitis [3]. The most common cause of bacteremia is cellulitis [4]. Invasive SDSE infections are becoming increasingly more common worldwide [2]. The invasive form of SDSE infection is more common among older patients especially, in the presence of underlying disease and in cases where skin integrity is disrupted. We report here a case of neonatal sepsis and meningitis caused by SDSE occurring 2 days after birth. We also reviewed the literature on SDSE infections.

Case report

The neonate described here is a male delivered to a gravida 1 mother vaginally at 38-weeks gestation with a birth weight of 3800 gr. The woman had rupture of membranes 8 hours prior to delivery. The rupture of membranes was spontaneous. The Apgar scores at 1 and 5 minutes were 8 and 9, respectively. The neonate began vomiting, developed cyanosis, had moaning and feeding intolerance beginning on postnatal 16. hours. While he was treated (only with ampicillin) in another hospital with the diagnosis of early onset neonatal sepsis, his general health state deteriorated so much on the postnatal 3. day, and he was referred to our neonatal intensive care unit (NICU) with fever, respiratory distress, seizures, peripheral cyanosis, and somnolence. On admission to our NICU, he was treated with oxygen therapy, intravenous fluid and dobutamine (5 µg/kg/min) for hypotension (arterial blood pressure of 23/14 mm Hg). An arterial blood gas showed metabolic acidosis (pH: 7.28, HCO3: 17, pCO2: 38). A complete blood count showed leukopenia (300 cells/µL), mild thrombocytopenia (130,000/µL) and an elevated CRP level (35 mg/dl) and elevated procalcitonin level (36 ng/ ml; normal range is <2 ng/dl) were detected. Renal and hepatic function test results (urea, creatinine, AST, ALT) and serum electrolyte levels were within normal limits. A lumbar puncture was performed, and analysis of CSF revealed the following values: WBC count 346 cells/mm3 (75% segmented neutrophils and 25% lymphocytes). The CSF protein level was elevated (650 mg/dl), and the CSF glucose level was decreased (21 mg/dl). Cultures of the blood, urine, spinal fluid, throat, external ear canal and rectum were obtained, and then antibiotic therapy was initiated (vancomycin, ampicillin, and amikacin). SDSE grew out of the CSF and blood cultures obtained on admission to the ICU. The microorganism was sensitive to penicillin G, cefotaxime, meropenem, linezolid and vancomycin and resistant to erythromycin, clindamycin, and levofloxacin (Minimal inhibitor concentration with E-test method=MIC values; penicillin G <0.12 mcg/mL, vancomycin 1mcg/mL). On the third day of therapy, a repeat lumbar puncture was performed. The CSF was clear,

hypocellular, the protein was 120 mg/dl, and the CSF glucose/ blood glucose ratio was 48/89. His mother did not allow us to obtain vaginal or anal swab specimens for antibiotic susceptibility testing. By 3rd weeks of treatment, the blood and CSF results were normal, and the child was clinically improved. The child's immunity was evaluated and found to be normal. Six months later there were no residual neurological sequelae.

Discussion

SDSE is a microorganism with increasing clinical significance, which causes both invasive and non-invasive infections [5]. Invasive infection with SDSE is seen in older individuals with a suppressed immune system or disrupted skin integrity. Group C β-hemolytic streptococcus have been involved in several localized human infections including pharyngitis, pyodermitis, cellulitis, wound infections, abscesses, erysipelas and necrotizing fasciitis. Severe invasive infections often occur in predisposed hosts: in fact they are common in patients affected by underlying immunodeficiency predisposing diseases or conditions such as age (neonate or elderly), diabetes mellitus, HIV-1 disease, alcoholism and injection drug use, and also in patients with chronic cardiovascular diseases and those undergoing chemotherapy or affected by cancer [6]. We can say that our case is in the immunodeficiency category because it is a newborn. Yamaoka et al. reported a case of streptococcal toxic shock syndrome caused by SDSE in a 12-hour-old newborn without a previous history of premature rupture of membranes or meconium aspiration [2]. In the literature, two cases of early onset neonatal sepsis caused by group G streptococci were reported before identification of this microorganism. One of these cases had a history of meconium aspiration, and the other had prolonged rupture of membranes [7]. The demographic and clinical characteristics of a total of 3 cases cited in the literature are shown in Table 1. The reported neonates -including ours- were delivered vaginally and had no identifiable risk factors.

In the literature, screening of pregnant women was recommended as for the presence of GBS to prevent the development of early-onset neonatal sepsis [8]. In our case, the mother did not undergo screening during pregnancy.

Analysis of the *emm* gene for the amino acid sequence situated and the N-terminal end of the M-protein in patients with SDSE has been performed in epidemiological studies related to epidemics caused by invasive and non-invasive microorganisms [2]. The presence of the *emm I* gene in children is associated with invasive infection.

SDSE cases reported in the literature are frequently older patients with an underlying disease. Mortality rates have been high among patients with SDSE. In patients with bacteremia due to SDSE, mortality rates are reported to be 15-18% [4]. In addition to this reported case of SDSE in a neonate at our hospital, only 2 other cases among neonates have been reported to survive. The prognosis of neonates with SDSE infection is better than adults. This may be because neonates usually do not have any underlying disease but adults with SDSE often do, and neonates may be diagnosed earlier while they are still in the hospital at birth.

In conclusion, SDSE may cause serious neonatal infections.

Table 1. Demographic and clinical	characteristics of Streptococcus	s dysgalactiae subspecies	eauisimilis	infection in neonatal cases.

References	Age	Gender	Delivery method	Birth weight (gr)	Clinical syndrome	Maternal screening (postpartum)	Underlying condition(s)	Therapy	Outcome
<u>Yamaoka et al,</u> 2010²	<5	Male	Vaginal delivery	2894	Streptococcal toxic shock syndrome, septicemia, meningitis	Positive	No	Ampicillin	Complete resolution
<u>Carstensen et</u> <u>al</u> , 1988 ⁷	<5	Male	Vaginal delivery	4000	Septicemia	Positive	Meconium aspiration	Penicillin Gentamicin	Complete resolution
<u>Carstensen et</u> <u>al</u> , 1988 ⁷	<5	Female	Vaginal delivery	3570	Septicemia	Result unshared	Rupture of membranes	Penicillin Gentamicin	Complete resolution
Present case	<5	Male	Vaginal delivery	3800	Septicemia, meningitis	Could not be performed	None	Ampicillin Vancomycin	Complete resolution

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

Conflict of interest

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

References

1. Puopolo KM, Madoff LC, Eichenwald EC. Early-onset group B streptococcal disease in the era of maternal screening. Pediatrics. Pediatrics. 2005;115(5):1240-6. 2. Yamaoka S, Ogihara T, Yasui M, Hasegawa M, Hira S, Oue S, et al. Neonatal streptococcal toxic shock syndrome caused by Streptococcus dysgalactiae subsp. equisimilis. Pediatr Infect Dis J. 2010;29(10):979-81.

3. Takahashi T, Ubukata K, Watanabe H. Invasive infection caused by Streptococcus dysgalactiae subs. equisimilis: characteristics of strains and clinical features. J Infect Chemother. 2011;17(1):1-10.

4. Rantala S. Streptococcus dysgalactiae subsp. equisimilis bacteremia: an emerging infection. Eur J Clin Microbiol Infect Dis. 2014;33(8):1303-10.

5. Anantha RV, Kasper KJ, Patterson KG, Zeppa JJ, Delport J, McCormick JK. Fournier's gangrene of the penis caused by Streptococcus dysgalactiae subspecies equisimilis: case report and incidence study in a tertiary-care hospital. BMC Infect Dis. 2013;20(13):381.

6. Milito C, Carello R, Fantauzzi A, Vullo V, Mezzaroma I. Streptococcus dysgalactiae subspecies equisimilis bacteremia in an HIV-1 patient with HBV/HCV co-infections: case report and literature review. Infez Med. 2014;22(3):241-6.

7. Carstensen H, Pers C, Pryds O. Group G streptococcal neonatal septicemia: two case reports and a brief review of literature. Scand J Infect Dis. 1988;20(4):407-10.

8. Verani JR, McGee L, <u>Schrag SJ</u>. Prevention of perinatal group B streptococcal disease revised guidelines from CDC. MMWR Recomm Rep. 2010;19(59):1-36.

How to cite this article:

Tarakci N, Yılmaz FH, Gültekin ND, Altunhan H, Örs R. Lneonatal meningitis due to streptoccocus dysgalactiae subspecies equisimilis: a case report and literature review. J Clin Anal Med 2018;9(3): 254-6.